A CURRENT OVERVIEW OF THE OCCURANCE, TOXICITY, AND DISPOSAL OF 2,3,7,8 - TETRACHLORODIBENZO -P- DIOXIN

2,3,7,8 - Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) is one of 22 isomers of tetrachlorodibenzo-p-dioxin (TCDD). As such it is also the most toxic of the 22 isomers. Both 2,3,7,8-TCDD and the other 21 TCDD isomers belong to a more general class of chlorinated dioxin compounds refered to as polychlorinated dibenzo-p-dioxins (PCDDs). Concerning the chemical characteristics of 2,3,7,8-TCDD , it is a non polar water insoluable molecule found to be a solid having a melting point of 305 ^{o}C. Furthermore, it is highly stable, being unrecative with strong mineral acids.

TCDDs, and in particular 2,3,7,8-TCDD, have entered the environment through a variety of pathways. Research has shown them to be present as trace contaminants in the manufacture of commercial chlorophenols and chlorobenzenes [1,3,32]. As such and through the use of chlorophenols as anti-fungal/anti-microbial agents, and also as intermediates in the manufacture of more useful chemicals 2,3,7,8-TCDD has entered the environment [3,9]. Furtheremore, 2,3,7,8-TCDD, in addition to PCDDs, has been found in both the particulate and gaseous emissions of a varied array of different combustion sources [1,2,4,5,6,7,8,10,33,34]. Thus, it has lead some researchers to state that 2,3,7,8-TCDD, as well as PCDDs, is ubiquitous in the environment [6].

The Occurrence of 2,3,7,8-TCDD in Commercial Products

It is recognized that 2,3,7,8-TCDD and many other PCDDs are formed as unwanted by-products in the manufacture of chlorinated phenols. Due to their extensive use either as intermediates, as fungicides, mold inhibitors, disinfectants, antiseptics, or wood preservatives chlorophenols are the largest source of PCDDs [3]. It has been estimated that present world production of chlorinated phenols is about 150,000 tons annually [1]. The most important chlorophenol intermediate is 2,4,5-trichlorophenol and its sodium salt 2,4,5-trichlorophenate. Both are used as starting materials in the production of the phenoxy herbicides 2,4,5-T [2] and 2,4-D, the bactericide hexachlorophen, and the diphenyl ether herbicides CNP, NIP, and X-52. All these compounds have been found to contain various levels of 2,3,7,8-TCDD and other PCDDs, which was the result of 2,4,5-trichlorophenol contaminated with PCDDs [3,35]. In addition to these compounds and the chemical wastes generated in their manufacture, the process equipment used in the manufacturing process can also be a source of 2,3,7,8-TCDD contamination. Specifically, process equipment will have been contaminated by 2,3,7,8-TCDD present in raw materials, finished products and wastes. This can thus result in the contamination of non 2,3,7,8-TCDD containing materials using the same process equipment if such equipment is not first decontaminated properly.

The Occurrence of 2,3,7,8-TCDD During Combustion

In addition to concerns that dioxins may be formed as unwanted by-products in the manufacture of chlorinated phenols and polychlorinated benzenes research over the past 10 years has shown that 2,3,7,8-TCDD, as well as PCDDs, also occurs in the combustion process. Olie, Huntzinger, and Karasek have found 2,3,7,8-TCDD, in addition to other numerous PCDDs, present in particulate form from many different types of combustion sources [4,5]. Their research indicates that 2,3,7,8-TCDD and other chlorinated dioxins occur in the particulate matter originating from the combustion of many different types of organic matter. Bumb stated that this was due to "a natural phenomena - the trace chemistry of fire which consists of numerous chemical reactions occuring during combustion" at concentrations as low as 10^{-10} percent"[6]. Additionally, other chlorinated compounds related to 2,3,7,8-TCDD and PCDDs such as polychlorinated terphenyls, chlorinated dibenzofurans,and polychlorinated biphenyl homologs such as decachlorinated biphenyls have also been found arising from combustion sources [6,7]. Evidence of the occurrence of chlorinated dioxins, including 2,3,7,8-TCDD, from the incineration of municipal solid wastes, sewage sludge, hazardous wastes, from internal combustion engines, as well as from the burning of fossil fuels and wood has been found by numerous researchers [2,4,5,6,7,8]. Of particular concern is the incineration of both hazardous wastes and municipal wastes.

It is well documented that improperly operated hazardous waste incinerators burning chlorinated organics or PCB laden wastes can result in the formation of 2,3,7,8-TCDD [6,9]. Regarding the incineration of municipal solid wastes 2,3,7,8-TCDD has been found in the precipitated fly ash and in small diameter air particulates which escape electrostatic precipitation [8]. Furthermore, the presence of all PCDD isomers has been found in the fly ash and air particulates eminating from municipal incinerators regardless of the type of refuse burned, the design of the incinerator, or the detailed composition of the organic adsorbed by the fly ash [5,10].

The chemistry and thermodynamics of the combustion process is very complex. Initial pyrolysis products formed during combustion involve hundreds of trace level compounds. These "trace combustion products result from the trace reaction chemistries of combustion, involving at least pyrolysis, oxidation, reduction, hydrolysis, and acidolysis, which call into play an assortment of interactions of ions - both atomic and molecular, electrons, free radicals, and free atoms" [6]. Due to these complexities associated with the thermodynamics and kinetics of the combustion process as well as the diverse makeup of municipal solid waste feed stocks ideal conditions exist for the formation of individual dioxins in amounts as high as 100 nanograms per gram of fly ash [6,7]. Not only is municipal solid waste an extremely complex mixture buts so are hazardous wastes, sewage sludges and fossil fuels. All are composed of literally hundreds of chemicals and compounds in varing amounts and concentrations

[6]. With this in mind researchers have analyzed fly ash eminating from incinerators. Their findings show over 400 compounds to be present (Table 1) and include polychlorinated benzenes, polycyclic aromatic hydrocarbons, and chlorinated phenols [7]. Additional research has shown that inorganic acid gases such as hydrogen chloride or chlorine gases evolved during the combustion process can cause the formation of PCDDs, including 2,3,7,8-TCDD, even in the burning of such fossil fuels as coal [11]. This has broad implications in that not only should the concentration of organochlorine compounds in incinerator feed stocks be of concern but also inorganic chlorine compounds should also be of concern.

Animal Toxicity

Paracelsus, an early 14th century "toxicologist" stated "All substances are poisons; there is none which is not a poison. The right dose differentiates a poison from a remedy". Fortunately, this is not exactly true. That is to say not every thing is generically toxic. However, for those substance that do produce a toxic response,2,3,7,8-TCDD included, care must be taken in extrapolating animal data to humans. One must realize that there are some uncertainties in biology which exist. That is, there is a fair amount of inter-species variation - man is not just a big rat - the metabolic pathways may vary from species to species. Also, that there is a fair amount of intra-species variation - among the different sexes of a species there can be a variation in the rates of metabolism or even variation within a sex.

When animal studies are used to extrapolate toxicological parameters to humans, the issue invariably raised is "which humans"? Mankind is highly diverse - genetically, culturally, and developmentally - with markedly differential susceptibility to pollutant induced adverse health effects. No one animal model can predict the responses of such a diverse grouping as humans. However, recognition of the diversity of potential models suggests that from this vast array of animals different biological systems could be found which reasonably simulate selected human groups. Many biological parameters such as enzyme activity levels, dermal thickness, repair processes, and blood variants display such a range that it may be possible to find one or more species with biological system(s) that respond similarly to their corrosponding human counterparts.

Numerous animal studies have been conducted on 2,3,7,8-tetrachlorodibenzo-p-dioxin using different species having a specifically similar target biological system(s) to that of humans. The most common indicator used in these studies has been the the median lethal dose or LD50,administered orally. The guinea pig has an LD50 ranging from 0.60 ug/kg to 2.1 ug/kg [12,13,14]. It is from these results that the 1 ug/kg lethality of 2,3,7,8-TCDD is taken [15]. Although this lethality is real for the guinea pig, its use as the sole toxicity indicator is in error since the guinea pig is hypersensitive to 2,3,7,8-TCDD and should not be used . Looking at other species (Table 2) we find that the female rat has an LD50 of 45 ug/kg while the male rat has an LD50 of 22.5 ug/kg [14]. Depending upon the species mice have an LD50 from 200 ug/kg to 300

ug/kg [13]. The rabbit has an LD50 of 115 ug/kg [13]. The monkey has an LD50 of < 70 ug/kg [13]. Further, hamsters have an LD50 of 5051 ug/kg showing the greatest resistance to 2,3,7,8-TCDD [16].

In determining the chronic or long term effects of 2,3,7,8-TCDD on animals multiple low concentration sublethal doses are administered. From such studies it has been observed that 2,3,7,8-TCDD is highly lipophilic thus depositing in such organs as the liver and adipose tissue as well as maintaining an equilibrium concentration in the blood (Tables 3, 4) [2,3,12,13]. As regards genotoxicity, 2,3,7,8-TCDD is not a mutagene but is a powerful teratogen and embryotoxin [12,13]. The embryotoxicity results from 2,3,7,8-TCDD's ability to pass through the placental barrier, which is in part a function of its lipophilicity [17]. In the process of carcinogenesis 2,3,7,8-TCDD functions more as a promoter than an initiator of the process [18]. 2,3,7,8-TCDD also results in damage to the immune system of animals even at very low doses, with a cumulative effect, resulting in the reduction of primary and secondary antibody production [19]. Furthermore, animal toxicology data shows that 2,3,7,8-TCDD results in liver and kidney degeneration, wasting away syndrome, damage to the immune system, and reproductive damage as the main chronic effects to animals.

Human Toxicity

Although extensive animal toxicological data on 2,3,7,8-TCDD exists it can be extremely difficult to fully extrapolate this data to humans as

a result of the high degree of biological variability present in the animal models used. However, in this particular case full extrapolation is not necessary. From the mid 1930s to the present over 24 industrial accidents, worldwide, in chemical plants manufacturing chlorinated phenols, have occurred resulting in over 1200 people being potentially exposed (Table 5) to 2,3,7,8-TCDD [20,21,22]. As a consequence a variety of clinical conditions have been observed in these exposed workers. These clinical conditions in conjunction with the available animal data aid in assessing the effects of exposure to 2,3,7,8-TCDD by man. The disorders that have been observed in these exposed workers include skin disorders, systemic effects, neurological disorders, and psychological disorders. The type of disorder and its extent vary depending upon the level of exposure to 2,3,7,8-TCDD. Exposure to 2,3,7,8-TCDD in the range of >1 ppm to 6ppm is characterized by the disorder chloracne [20,21,22,23]. This is accompanied by impaired liver function, which is reversible, or liver damage, kidney damage, pancreatic damage, cardiovascular disorders, elevated serum lipids, gastrointestinal disorders, myopathy and neuropathy, including depression and irritation of the central nervous system (Table 6). As concerns low level exposure to 2,3,7,8-TCDD, 1 ppm to several hundred ppb, generally no chloracne results. Additionally, all the above disorders appear, although more mild in their manifestation. An exception to the above disorders is that no liver and kideny damage results. Instead impaired liver and kidney function result [21,22,23]. Furthermore, these disorders are not progressive and disappears with time

[21,22,23]. However, chronic very low level exposure, in the part per billion to tens of parts per billion range, should be of concern as elevated serum lipids can lead to cardiovascular disease such as heart attacks. At extremely high levels of exposure, much greater than 6 ppm, to 2,3,7,8-TCDD, for example by dermal contact, extensive liver, kidney, and pancreatic damage will result leading to death. This has occurred on two separate occasions with two fatalities [20].

Concerning the human teratogenic effects of 2,3,7,8-TCDD, it has been shown that 2,3,7,8-TCDD is not a male teratogen [21,22]. However, sufficient human information is not available to draw a conclusion as to whether it is a female teratogen and embryotoxin, although animal studies indicate that it is. As regards the carcinogenic effects of 2,3,7,8-TCDD, conflicting evidence exists as to whether 2,3,7,8-TCDD exposure results in the formation of soft tissue sarcoma. However, general indications are that 2,3,7,8-TCDD exposure at very low levels does not result in carcinogenesis taken place [21,22,23].

Thus in general, it is unlikely that permanent, severe, and debilitating disorders are inevitable after exposure to 2,3,7,8-TCDD not sufficient to produce chloracne. However, it must be remembered that individual susceptibility due to hypersensitivity may make some people more vulnerable to the effects of 2,3,7,8-TCDD [22].

The Handling of 2,3,7,8-TCDD Contaminated Materials

Whether the source of 2,3,7,8-TCDD or PCDD contaminated material is

fly ash from municipal solid waste incinerators or waste residues from chemical manufacturing, care must be taken to limit human exposure during disposal handling. To do so not only requires that workers handling such wastes be provided with the proper personnel protective equipment but that they also be trained in (a) the hazards associated with the material, (b) the potential routes of entry into the body by that material, (c) proper handling of the material, (d) the proper selection and use of protective clothing and equipment, and (e) the importance of personal hygiene after removal of protective equipment, i.e. showering [24,25]. Workers involved with the disposal of dioxin contaminated materials should be equipped with class C protection [29]. This involves the use of full body chemically resistant suits with full head and neck covering, a full face cartridge type chemical respirator, chemically resistant gloves, and neoprene safety boots. Additionally, workers should also be provided with both undergarments and overgarments which can be disposed of after each use along with all used protective clothing and equipment. Furthermore, separate areas for changing and showering should be provided. With this there must be four distinct areas: (1) a "clean room" where street clothing is removed and put on, (2) a shower room where employees shower after the removal of protective equipment but prior to wearing street clothing, (3) an isolation room where employees wearing protective equipment after exposure are decontaminated, and (4) a dressing room where employees don and remove supplied undergarments, overgarments, and protective clothing and equipment. Finally, all employees handling such

wastes should be given extensive "baseline" physicals prior to beginning such work by an occupational physician, with particular attention given to liver function, kidney function, and skin condition. These physicals should be followed semiannually for a minimum of one year after removal from this type of work environment.

Disposal of 2,3,7,8-TCDD Contaminated Materials

In disposing of 2,3,7,8-TCDD or any other PCDD contaminated materials three potential methods exist; (1) entombment, (2) photolytic decomposition, and (3) incineration [26]. The use of entombment is a short term solution that serves as an effective tool in the safe handling and storage of 2,3,7,8-TCDD contaminated material while a more permanent solution to the problem can be researched and implemented. Entombment involves the construction and use of an above ground or below ground reinforced concrete monolithic structure storage facility that can be sealed or capped initially and reopened later on. Before being placed into this structure all 2,3,7,8-TCDD contaminated material is collected and containerized, for example in 55 gallon storage drums, properly labeled, and then sealed. The then properly contained material is placed into the structure. After the entombment facility is completely filled it is sealed until a suitable solution is found. However, the facility must be maintained under constant monitoring to detect any potential leaks either from the housed contained waste or from the facility to the outside environment. One potentially significant problem with this short term

solution is that once the waste material is finally disposed of, the facility itself may have to be dismantled and disposed of as well if it has been contaminated by the stored 2,3,7,8-TCDD waste, hence increasing the total amount of material to be disposed of.

Photolytic decomposition or incineration, by virtue of being "destructive modes of disposal, can be viewed as ultimate solutions in the disposal of 2,3,7,8-TCDD contaminated materials. By stating that these two disposal methods are destructive it is inferred that they result in the irreversible decomposition of a chemical contaminant. For photolytic decomposition to be used the 2,3,7,8-TCDD contaminated material must first be extracted with a suitable solvent such as hexane. After a suitable extraction of 2,3,7,8-TCDD, > 99.9% efficiency, a small amount of isopropanol is added to serve as an additional source of hydrogen donors. This final extract is then passed through a photo-oxidative reaction chamber where UV light having an emissive wavelength from 200nm to 320nm and a radiant intensisty of $2mW/cm^2$ bombards the solution [30,31]. This results in the complete photolytic decomposition of 2,3,7,8-TCDD - dechlorination followed by complete polymerization of photoreductive decomposition by-products of 2,3,7,8-TCDD [24,27].

With incineration the material can be introduced into the incinerator either directly in unextracted or extracted form [26,27]. For 2,3,7,8-TCDD materials that must be extracted, the same procedure used for photolytic decomposition is followed, with the exception that isopropanol is not added. The material is then introduced into a two chamber

incinerator where it is thermally oxidized. In the primary incinerator chamber the waste is pyrolysied at a temperature of 1600 ^{o}F. The material vaporized during this step is then swept into the second chamber. This second chamber, acting as an after burner, completely oxidizes any 2,3,7,8-TCDD present at a temperature of 2300 ^{o}F and in the presence of excess air , with a residence time of at least 2.2 seconds [26]. This type of incinerator design has interesting possibilities when applied to the incineration of municipal solid waste. By the use of a high temperature after burner with excess air all combustion by-products including dioxins should be completely destroyed.

Conclusion

2,3,7,8-tetrachlorodibenzo-p-dioxin occurs as a trace contaminant in many chlorophenols and their respective waste streams. However, this is not the only source. The incineration of municipal solid waste as well as the burning of fossil fuels produces combustion by products which include a number of dioxins including 2,3,7,8-TCDD. The formation of dioxins by combustion thus involves the complex processes of combustion thermodynamics and kinetics whereby combustion by-products such as polycyclic organic matter and acid gases such as hydrogen chloride inter-react to form chlorinated cyclic and polycyclic organics including 2,3,7,8-TCDD.

Concerning the toxicity of 2,3,7,8-TCDD, numerous animal studies have been conducted using a variety of species. It is by one of these

species, the guinea pig, which is hypersensitive to 2,3,7,8-TCDD, that dioxin has earned the ominous connotation "the most toxic synthetic chemical known to man". Although 2,3,7,8-TCDD is highly toxic, man tends to show a greater resistance to its effects than many other species. With over 1200 people having been exposed to 2,3,7,8-TCDD worldwide from industrial accidents involving the manufacture of chlorophenols only two fatalities have been report to have resulted from 2,3,7,8-TCDD exposure. However, further research is necessary to fully understand the long term health effects of 2,3,7,8-TCDD at both low and high level sublethal exposures.

In handling 2,3,7,8-TCDD contaminated materials for disposal proper care must be exercised. Proper protective equipment and clothing must be used as well as effective training in the use of such equipment. Additionally, the potential hazards and possible routes of entry associated with 2,3,7,8-TCDD must also be fully understood.

Finally, if ultimate disposal is sought the 2,3,7,8TCDD contaminated material should either be destroyed by extraction-photolytic decomposition or incineration. However, if the waste or material matrix is so complex that this is not presently technically feasible the waste should be entombed in a special storage facility until a suitable ultimate disposal method is found. Also, in the incineration of municipal solid waste the use of a dual chamber incinerator with a primary chamber having a temperature of 1600 ^{o}F and the secondary chamber having a temperature of 2300 ^{o}F should result in no PCDDs, including 2,3,7,8-TCDD, being formed through combustion by-product inter-reactions.

TABLE 1

Some Selected Polycyclic Organic Compounds Found In Effluent Gases From Coal And Refuse Combustion.

COMPOUND	AMOUNT (ug/dcm)
Napthalene	1.7
Acenaphthylene	1.3
Fluorene	0.13
Phenanthrene	2.0
Pyrene	0.06
Benzo (a) Pyrene	0.001
Dimethyl Phthalate	0.07
Diethyl Phthalate	5.7
Di-n-butyl Phthalate	14.0
Butyl benzyl Phthalate	1.1
Bis (2-ethylhexyl) Phthalate	13.0
Di-n-octyl Phthalate	2.1
TetrachlorobiPhenyl	0.59
PentachlorobiPhenyl	1.86
HexaclorobiPhenyl	1.09
HePtachlorobiPhenyl	1.13
OctachlorobiPhenyl	0.17
1,4-Dichlorobenzene	74.0
1,2-Dichlorobenzene	700
1,2,4-Trichlorobenzene	15.0
1,2,3-Trichlorobenzene	7.0
1,2,4,5-Tetrachlorobenzene	0.6
1,2,3,4-Tetrachlorobenzene	1.0
Pentachlorobenzene	0.8
Hexachlorobenzene	0.3

Note: ug/dcm = mircogram per dry cubic meter.

Excerpted from reference 28.

TABLE 2

COMPARISON OF ORAL LD50 FOR 2,3,7,8-TCDD

SPECIES	LD50	
Rat(male)	22.5	ug/Kg
Rat(Female)	45	ug/Kg
Mice	200-300	ug/Kg
Guinea Pig(male)	0.6	ug/Kg
Rabbit	275	ug/Kg
Monkey	50-70	ug/Kg
Hamster	5051	ug/Kg

TABLE 3

GASTOINTESTINAL ABSORPTION OF 2,3,7,8-TCDD

SPECIES	VEHICLE	DOSE SCHEDULE (ug/Kg)	% Absorption Mean +/- SD
Guinea Pig	-	Single dose	50
Rat	7PPb in diet	0.5ug/Kg/day X 42 days	50 - 60
Rat	20PPb in diet	1.4ug/Kg/day X 42 days	50 - 60
Hamster	Olive Oil gavage	650 ug/Kg Single Dose	74+/-23

TABLE 4

DISTRIBUTION OF 2,3,7,8-TCDD IN BODY ORGANS

SPECIES	ROUTE OF ADMINISTRATION	PRINCIPAL ORGAN DEPOTS
Guinea Pig	Oral	Fat>Liver>Adrenal >Thymus>Skin
Rat	Oral	Liver > Fat
Mouse	Oral	Liver>Fat>Kidney >Lung
Monkey	Intraperitoneal	Fat>Skin>Liver >Adrenals=Thymus
Hamster	Oral	Liver > Fat

TABLE 5

ACCIDENTS IN CHEMICAL PLANTS INVOLVING THE MANUFACTURE OF CHLOROPHENOLS AND RELATED OCCUPATIONAL EXPOSURE RESULTING IN HUMAN ILLNESS

DATE	COUNTRY	PRODUCT	PERSONNEL AFFECTED
1936	Michigan	TetraCP	21
1936	Mississippi	TetraCP	300
1949	West Virgina	2,4,5-T	228
1949	West Germany	TCP	17
1952	West Germany	TCP	31
1953	West Germany	TCP	55
1956	France	TCP	17
1956	New Jersey	2,4,5-T/2,4-D	29
1959	Italy	TCP	5
1963	Holland	2,4,5-T	106
1964	USSR	2,4,5-T	120
1964	Michigan	2,4,5-T	30
1965	Czechoslovakia	TCP	80
1968	UK	TCP	90
1969	Czechoslovakia	TCP	80
1970	UK	TCP	90
1970	Japan	PCP/2,4,5-T	25
1972	USSR	TCP	1
1972	Austria	2,4,5-T	50
1973	Austria	2,4,5-T	50
1974	West Germany	2,4,5-T	5
1976	Italy	TCP	134
1978	UK	TCP	41

Excerpted from references 20,21,and 22.

TABLE 6

CLINICAL PROBLEMS ASSOCIATED WITH TCDD EXPOSURE BY HUMANS

SKIN DISORDERS

Chloracne
Hyperpigmentation
Hirsutism

Psychological DISORDERS

Depression
Sleep distrubances
Personality Changes
Loss of drive and energy
Loss of Libido

SYSTEMIC EFFECTS

Fibrosis of liver
Increased SGOT
Increased SGPT
Cardiovascular disorder
Uninary tract disorders
Pancreatic Disorders
GI tract disorders
Weight loss
Anorexia
Depression of immune system
Elevated serum lipids
Muscular aches and pains

NEUROLOGICAL DISORDERS

Polyneuropathy
Lower extremety weakness
Sight Disturbances
Hearing Loss
Headaches
Loss of smell/taste

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